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MEDICAL ARTS AND SCIENCES

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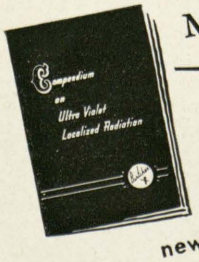

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EDITORIAL

CANCER OF THE LUNGS*

H. JAMES HARA, M.D.

Cancer of the lungs is definitely on the increase. It constitutes 10 per cent of all forms of malignant diseases. Chronic irritation appears to be one of the chief factors in its pathogenesis. Ninety per cent of a series of patients reported from the Cook County Hospital in 1936 were chronic smokers. In a series of 175 cases observed at the St. Luke Hospital and the Research and Educational Hospital, Chicago, in 1945, 50 were heavy smokers, 14 nonsmokers, and the remainder were noncommittal as to their smoking habits.

Exhaust gases from automobiles, tar on roads, repeated respiratory infections, and hazards from industrial dust and fumes have been suggested as predisposing factors in cancer of the lungs. So far nothing definite has been accepted as truly causative. It is a disease of white males of cancer age, though it is occasionally seen among the colored races and in those under twenty years of age.

The onset of bronchiogenic carcinoma is almost always insidious. Cough, sputum, and pain are the triad symptoms of early bronchiogenic carcinoma. The cough is invariably irritating and nonproductive at first. Those who smoke often attribute their cough to the irritating effect of tobacco on the mucous membrane of the respiratory tracts. Some try to reduce the quantity of their daily consumption of tobacco, others change the brand of cigarettes, and still others voluntarily give up smoking without any appreciable change. A much larger group consider the cough as resulting from a so-called cold or virus bronchitis, which fails to clear up as does their usual seasonal upper respiratory infection. Often cough recurs after subsiding for a week or two, and is interpreted by the patient to be a fresh head cold. Sputum of bronchiogenic carcinoma is scanty and mucoid at first. Later, when ulceration occurs, it gradually increases, becomes blood streaked and purulent.

Pain of bronchiogenic carcinoma is more or less a dull ache, always limited to the side affected, intermittent, and of short duration in the earlier periods. In some it appears as a substernal ache, radiating to the shoulders. "Tightness," a "bandlike constriction around the chest," "inability to fill the chest with each inspiration" are some of the expressions used to describe the thoracic discomfort. Symptoms are vague and intermittent at first, but later become more pronounced and continuous. The thoracic pain is aggravated by coughing. The patient holds his chest as he coughs, in order to lessen the sensation of discomfort. Pain always signifies spread of the growth. In patients with a peripheral growth pain appears early, often simulating pleurisy.

The complication of bronchiogenic carcinoma is chiefly concerned with obstruction of the respiratory tract, caused either by intra-

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* From the Department of Otolaryngology, College of Medical Evangelists.

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THE OTOLARYNGOLOGIC PATIENT AND NUTRITION*

LLOYD K. ROSENVOLD, M.D.

According to the National Research Council, it is a statistical fact that at least two out of three Americans are living on faulty diets. Different figures are given for various studies, and in a recent survey of school children in the State of Vermont (Pierce), certain signs of past or present inadequate nutrition were found in 85 per cent of the children. The physician who yesterday treated thirty average patients consequently saw twenty individuals who were in need of dietetic and nutritional guidance. When to this number are added those who use tobacco, alcohol, caffeinated drinks, and other drugs, to say nothing of their indulgence in irregular habits of living, there remain few patients indeed who would not benefit by sound advice on diet and living habits. It is obvious that responsibility for giving this advice must rest with each individual physician, be he a general practioner or a specialist; for consultation with a nutritionist is not available to, or practical for, the great majority of patients.

To sit down with each patient and verbally elicit a nutritional history is not practical and consumes too much time. To circumvent that difficulty, I use a four-page questionnaire** in fine print, which is given to the patient on the occasion of his first office visit and returned at or before the second visit. The answers can be quickly surveyed and summarized, and thus one may gain a clear picture of the individual's nutritional and living habits, as well as symptoms pertaining to the nutritional and metabolic state. The findings from these questionnaires have been very revealing to me, as well as to the patients, for some of them have not realized how irregular their own ways of living were, until they summarized them on paper.

One of the outstanding nutritional errors of Americans is their excessive use of cane sugar, which is an excellent source of caloric energy but lacks minerals and vitamins. During the year 1944, when sugar was seemingly strictly rationed, the average American consumed 104 grams daily (Gubin), and in 1939 approximately half of the daily caloric intake of the average American consisted of refined sugar and white wheat flour (Cowgill). The latter food is also deficient in vitamins. Not only does sugar lack vitamins, but in the metabolic process of using the sugar the available supply of vitamins of the B group is quickly used up, and a vitamin deficiency state may ensue. This was dramatically illustrated in the recent case of a young lady of good family, living on an essentially normal diet, except for

* From the Department of Otolaryngology, College of Medical Evangelists.

** The space allotted to this article does not permit publication of the questionnaire or details concerning its use. Copies of the form are available on request. Similar history forms have been described in the literature by Moose, Roberts, and Spies.

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the fact that she worked at a soda fountain and partook heavily of cane sugar in the form of ice cream, milk shakes, and sweetened drinks. She presented a typical picture of riboflavin deficiency with glossitis and fissuring at the angles of the mouth. In addition she had been suffering with asthma and hay fever for several years. Oral riboflavin therapy caused prompt healing of the mouth lesions, and additional nutritional guidance also effected relief of the hay fever symptoms.

Vitamins have lost a degree of their former mystery, and we now know that some of them are intimately associated with the enzyme systems in body metabolism. For example, thiamin aids in cellular oxygen uptake, and it has been demonstrated in pigeons that when it is deficient, pyruvic acid will accumulate in brain cells. In humans there is also an increase in blood and tissue pyruvate (*Nutrition Reviews*). If thiamin deficiency produces such profound changes, and riboflavin deficiency can produce ulceration of the lip margins, it is quite likely that a grossly deficient diet will produce other severe changes in the tissues of the ear, nose, and throat. It is to be hoped that eventually these effects can be better demonstrated and classified.

Much has been said and written in recent years about the importance of psychosomatic factors in aggravating the symptoms of many patients. Recent reports (Williams *et al.;* Keys *et al.*) have shown that certain of the symptoms of neurosis can be produced experimentally by diets deficient in some of the B vitamins. Thus when evaluating a patient's complaints we should also evaluate his diet, for some of his feelings may be due merely to impaired nutrition and not to organic disease or a functional neurosis.

It is not my purpose to infer that one group of food factors, such as the vitamins, is the all-important thing, for in recent years the role of proteins in metabolism has also received special attention, as is attested by the increased use and study of amino-acid preparations, and we are now beginning to understand the function of some of the individual amino acids. A diet history form enables the practitioner quickly to determine whether the patient receives too much or too little protein and the approximate biologic value of each type. Mineral intake may also be readily evaluated.

Another common American evil is self-medication, and of this the laxative habit is perhaps the foremost. Laxatives constitute a definite nutritional hazard in that many times they interfere with digestion and absorption of certain essential foodstuffs, the most classic example, of course, being the effect of liquid petrolatum in preventing absorption of fat-soluble vitamin A. In many cases advice about simple hygienic habits to replace the need for cathartic drugs will be very successful, and it is doubtful that specialists in colon diseases will object to this advice being given, for the otolaryngologist is not treating colon disease but is merely advising the patient to abandon an unnecessary, health-destroying habit. Salicylates and sulfonamides, besides their direct toxic action on tissues, may also interfere with vitamin metabolism.

Allergic individuals have been found to benefit by nutritional correction. I do not refer to elimination of allergenic foods but to correcting the entire dietary so that the body has opportunity for normal nutrition. The idea that nutritional deficiency or imbalance might be a cause for an allergic state is not new, for Russell Wilder has stated, "Yet some evidence suggests that allergy itself may be occasioned by unbalanced diets." The nutritional approach to treatment of allergic states will be reported in a separate study, so no further mention will be made now.

I have found patients who have been placed by physicians on very restricted diets or potent

drugs months or years before, and even after the need for the diet or treatment may have expired, the patient continues the program. This is not uncommon, and unless a careful history is elicited, the fact may be overlooked. Spies has shown that so-called special diet programs, which we as physicians have used for many years, are often deficient diet programs.

Not only is the nutritional history idea helpful to the physician, but the patients are usually gratified and pleased that their doctor will take such a detailed interest in their health. It can be adapted to almost any field of medical practice, and those who have used the plan have found it very helpful. I personally feel that if I should fail to consider my patients' nutrition carefully, I would often rob myself of helpful diagnostic criteria, and in turn inadvertently withhold from the patients essential help and advice. Otolaryngology has passed through several overlapping phases of therapy, usable features of each, of course, remaining to this day. The antiseptic era has come and gone. Then came the anatomical (surgical) era, to be followed by the immunologic period, and it, in turn, by the chemotherapeutic one. Now we are in the midst of the antibiotic age, but the nutritional era, which has waited in the background for many years, seems about to come of age.

SUMMARY

A brief survey has been presented showing the close relationship between faulty nutrition and some of the common conditions seen in otorhinolaryngologic practice. The benefits that accrue, both to the physician and to the patient, from a detailed nutritional history are stressed, and the suggestion is made that the elicitation of the history is facilitated by using a comprehensive, yet simple, printed form which the patient can fill out at home. The method is adaptable to fields of practice other than otorhinolaryngology.

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IRRADIATION OF THE NASOPHARYNX*

CHARLES E. FUTCH, M.D., and JOHN D. ABBEY, M.D.

Irradiation of the nasopharynx by the otolaryngologist is not longer in the experimental stage but is a definite and accepted therapeutic adjunct in the field of otolaryngology. Crowe with his associates, in otolaryngology, and Burnam, in radiology, both working at the Johns Hopkins Hospital for the past ten years, have in their experimental work shown conclusive evidence of the value of irradiation of the nasopharynx, particularly in patients showing an impairment of hearing for high tones.

The impaired hearing for high tones was universally thought to be due to an inner ear nerve lesion, and according to Crowe this was usually found to be true. In a few cases, however, he found the organ of Corti normal histologically and changes in the middle ear only were found, secondary, in his opinion, to an overgrowth of lymphoid tissue in and around the pharyngeal orifice of the eustachian tube.

It has been known for many years that the lymphocytes are extremely sensitive to irradiation, and Crowe turned to his associate, Burnam, for advice as to the best method of approaching the mass of lymphoid tissue in the nasopharynx. The radium applicator, as later described, was devised, and used successfully for the treatment of this lymphoid tissue, which was most difficult to remove surgically without causing, as a rule, severe cicatrization. Marked improvement followed irradiation of the orifice of the eustachian tube in these selected cases of chronic eustachitis, and middle-ear deafness was subsequently greatly improved.

The advantages of the application of radium directly to the orifice of the eustachian tube is obvious; namely, the radiation does not have to pass through innocent tissue and thus cause the inevitable changes that take place in all tissue following radiation. Further, the small dosage of radiation in the procedure recommended below may be applied from one to several times, with the results under the direct observation of the otolaryngologist. Burnam has said that the specialist in ophthalmology and otolaryngology should master the principles of ray theray, and either carry out the treatments personally or have these treatments carried out in the way that is likely to produce the best results.

The procedure of irradiation as advised by Crowe is as follows: Three treatments are usually given, each 8½ to 12 minutes to each side, with the 50 milligram monel metal radium applicator in direct contact with the tissue to be irradiated. An observation period of from thirty to sixty days after the third treatment is advised before further therapy. The object of irradiation, according to Crowe, is to remove the particular lymphoid tissue which is causing the patient's symptoms; namely, to reduce the lymphoid tissue at the orifice of the eustachian tubes and not to remove all the lymphoid tissue in nasopharynx. If the symptoms are relieved after three radiations, it is unnecessary to give further irradiation, but if needed, further treatment may be given, with proper control. During the war approximately

* From the Department of Otolaryngology, College of Medical Evangelists.

25,000 treatments were given with the monel radium applicator by medical officers of the Army Air Forces and at the New London submarine bases in order to prevent recurrent aerotitis. The average result was 90 per cent effective, and not a single instance of radium burn or poisoning or a drop in the white blood count has been reported. According to Crowe, the only requirements for procedure are an accurate examination with the nasopharyngoscope, location of the tissue to be treated, and placement of the applicator so that the side, not the end of the radium-containing chamber contacts the tissue observed. The time element, namely 8½ to 12 minutes, must be accurately observed, and this is best done with an automatic interval timer. The applicator as used has the radium filtered by one millimeter of monel metal; thus filtered, the beta rays, which do not penetrate as deeply as the gamma rays but are more effective on the lymphoid tissue than the gamma rays, are chiefly relied upon. The result of the irradiation is thought to be an inhibition of mitosis in the germinal centers of the lymphoid tissue; thus the new cells, lymphocytes, do not multiply, and the lymphoid mass is reduced as the old cells disappear. The use of radium in the nasopharynx is not indicated if the lymphoid mass is large. For this reason irradiation should follow an adenoidectomy rather than precede it.

Asthmatic children have, according to Crowe, frequently shown marked improvement following the second irradiation, but more usually about a month after the third irradiation. Exacerbation of the asthmatic attacks has frequently been observed by the authors up to sixty days.

Ward and Moffatt have reported a series of thirty-four asthmatic children in which all forms of asthma were represented; 68 per cent of the patients obtained from total to 50 per cent relief.

Some recurrence of adenoid tissue after surgery is so frequent that this recurrence must be regarded as almost normal. Thus the history of an adenoidectomy does not essentially mean that without the use of the nasopharyngoscope the nasopharynx may be dismissed as an etiologic factor in either deafness or asthma.

The recurrence of the lymphoid tissue itself, particularly in individuals of the lymphoid diathesis, is so usual that, as with the adenoids, some recurrence is usual. In our series of some four hundred irradiations of the nasopharynx, it was found that six months to a year is the usual time of relief relative to some recurrence of lymphoid tissue. However, the recurrence of the primary deafness or in the cases which have shown improvement relative to their asthma has not reappeared essentially at this time. Further radiation at the time of this recurrence of lymphoid tissue is, in our opinion, indicated, and for that reason it is emphasized that this technic be mastered by the otolaryngologist, so that the tissue may continuously remain under his observation.

In conclusion, in using the radium applicator it is essential to protect yourself and your office personnel, because of the repeated short exposures, which are, of course, cumulative. Care must be exercised both in the storage and in the therapeutic use of the radium.

SUMMARY

1. The use of the 50 milligram monel metal radium applicator for irradiation of the nasopharynx has been definitely established as a valuable therapeutic adjunct in the field of otolaryngology.

2. The specificity of action of irradiation upon lymphoid tissue causes this type of treatment to be indicated wherever masses of this tissue are thought to be factors in the hard of hearing or in asthmatics.

3. Irradiation of the nasopharynx can be

carried out by the otolaryngologist who has familiarized himself with the procedures or by the radiologist working with the otolaryngologist: it is advisable that the treated area remain under the observation of the physician who is acquainted with the normal appearance of the localized tissue.

4. The simplicity of procedure in itself recommends that all who treat either deafness or asthma at least be aware of the procedure.

5. Care should be exercised in the protection of the office personnel from the cumulative effect of short exposures to the radioactive substance.

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PLASTIC SURGERY OF THE NOSE AND FACE*

EDWARD KING, M.D.

Special knowledge and training are necessary for surgeons who plan to do plastic operations about the nose and face. A thorough knowledge of the external nose is essential. The physiology of the nose must be taken into consideration when planning operative procedures. Plastic surgeons are importuned constantly by patients to perform trivial corrections for imaginary defects. Experience is necessary to determine when a correction is indicated and how much it will accomplish. Because of the publicity which all types of surgery receive the layman is confused. Many laymen believe that skin grafting and removal of wrinkles and scars can be performed in a short session in a surgeon's office, much like a beauty treatment. The plastic surgeon must safeguard the interests of the patient and at the same time protect himself from criticism by explaining fully to each patient the nature of the operation, what it may accomplish, and the risks involved. A frank discussion with each patient is recommended, so that the patient with a slight defect may be persuaded to let well enough alone. When prospective candidates for plastic surgery are informed that the face-lifting operation is a hospital procedure of major importance, the determination fades rather promptly in most cases.

The indications for operation must be clear, and a fair chance of improvement is a prerequisite. Accurate records must be kept in order to evaluate results. We use photographs and casts previous to operation. The history is important in all cases, especially following trauma. Operative procedures must be carried out under strict asepsis, and therefore hospitalization is required. When possible, the patient should be hospitalized even for minor corrections, because in plastic surgery failure is especially tragic.

Injuries to the nose and face are so common as a result of auto accidents that special attention should be devoted to these patients in all hospitals. Most of the principles involved in handling these injuries are well known and hardly need further emphasis. It is well, however, to keep in mind the principles that underlie the proper handling of recent injuries. Sometimes the injuries to the nose and face are of minor importance at the time of the accident because of more serious injuries elsewhere. It must not be forgotten that deformities which result from facial injuries will assume great importance to the patient once recovery is in sight. Corrections of injuries should be attempted immediately where feasible, but when the patient is seen hours or days after the injury, there is so much swelling that it is difficult to estimate the extent of the injury. It is advisable then to wait until some of the swelling has subsided before making an attempt to correct it. Ordinarily the correction can be deferred for a week or ten days. There is always a chance that the healing may be rapid and solid, and the correction may not be possible after ten days or two weeks. It is my experience, however, that some of these cases can be corrected after three or four weeks by simple elevation and pressure, as is done in

* From the Department of Otolaryngology, College of Medical Evangelists.

any acute fracture. All wounds of the face must be repaired at once, after a thorough cleansing of the tissues. Special attention should be given to road dirt which becomes embedded in the skin and produces ugly pigmentation of the injured part. Its removal is accomplished by stiff brushing or even excision. Very fine sutures and needles should be employed in closing wounds of the face, and drainage must be provided when the wounds are deep.

Figure 1.—Saddle nose, result of injury.

Repair by narrowing bridge, cartilage implant, reduction of alar cartilages, and strut in columella.

SADDLE NOSE

Saddle nose deformity is most common following injuries to the nose. It should be pointed out that sinking in of the nasal dorsum almost invariably occurs where injuries of the nose are neglected. The injured nose should receive careful inspection inside and out, because a large hematoma of the septum may form following injury. Unless this is treated at once, infection follows, resulting in loss of septal support and a saddle deformity of nose. These deformities must be corrected by means of a cartilage implant (Figure 1.) Preserved cartilage has now been used successfully for a sufficient time to warrant its use in most cases.

TWISTED OR DEVIATED NOSE

As a rule this deformity is due to a dislocation of the septum. Its correction consists in a complete mobilization of the septum, with proper fixation of the mobilized septum in the midline. If the nasal bones and the nasal proc-

esses are deviated from the midline, they must be mobilized by fracturing and freeing them from their attachments and placing them in the midline. At times the septum is so badly twisted and macerated that it is advisable to remove it, straighten it, and replace it in the midline, supplementing the cartilage with a plate of preserved cartilage and a strut in the columella for tip support. As a rule, dislocation of the septal cartilage occurs in childhood, and the injury is overlooked, resulting in a deformity which is not noticeable until puberty. It should be emphasized that injuries to the nose in childhood should be inspected carefully for dislocation of the septum.

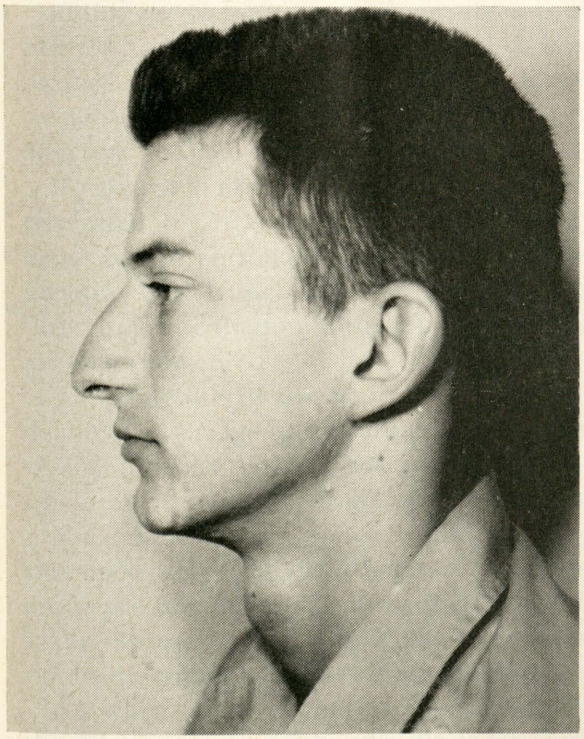
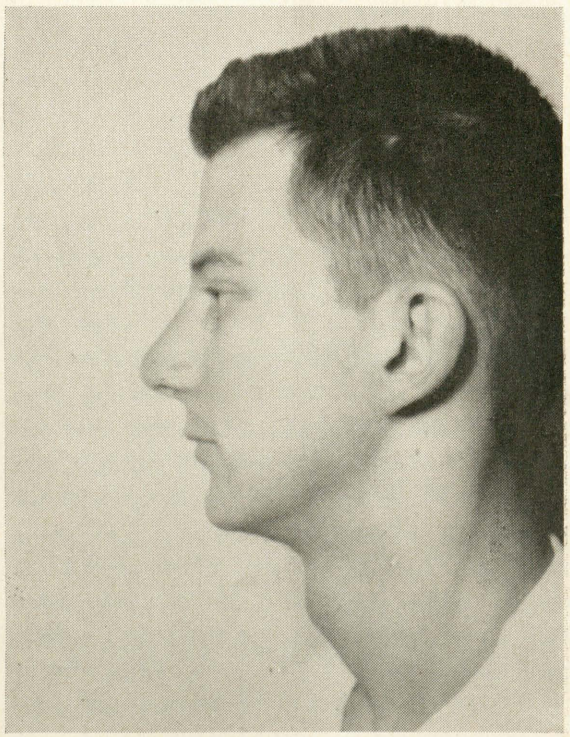


Figure 2.—Hump nose with large tip.



After removing hump and reducing tip.

LOSS OF SKIN

Where skin is lost, either on the nose or where it must be supplied, as in large defects of the nose or face, it is always best to use skin from some area above the clavicle in order that the color and texture may be maintained. Skin from the abdomen or thigh or arm is never satisfactory. For small defects the skin from behind the ear or the upper eyelid may be used as free full thickness grafts. For large losses, especially in women, the skin from the forehead may be employed as a flap.

CONGENITAL DEFORMITY

Congenital deformities make up a large portion of the cases that come to the attention of the plastic surgeon. Hump nose and other deformities must be corrected when they are conspicuous and a source of anguish to the patient.

As a rule the removal of a hump necessitates narrowing of the bridge and reduction of

the tip in order to make the nose conform to the face (Figure 2).

Removal of scars and wrinkles must be approached with care in order that the final result may be an improvement. Congenital deformities of the ears should be repaired before school age if it is possible for this to be done.

CONCLUSIONS

A thorough knowledge of anatomy and physiology of the nose is a requirement of the surgeon who plans to do rhinoplasty. In planning for an operation, the surgeon must keep in mind the end results expected by the patient. If the indications for the operation are well established, the results will be better.

FRACTURES OF THE NASAL AND THE MAXILLARY BONES*

MILO C. SCHROEDER, M.D.

In this age of rapid transit, with its unprecedented high accident rate, a structure occupying such an exposed position as the nose would seem to demand of the physician knowledge of both first aid and subsequent care.

If a fractured nose and maxilla are not properly cared for, the consequences to the patient's future personality may be serious and lifelong. Both functionally and esthetically, then, proper reduction of these bones is of major importance.

The structure of the nose is like that of a double-span bridge in which the displacement of any member distorts the position of the whole and alters the function of the unit.

Since the maxillary bones form the foundation upon which the nasal bones are attached, any depression or displacement there will generally distort the nasal bridge so that it cannot be functionally correct. This situation will occur when a heavy blow is received on the cheek and the nose. The maxilla is fractured through the infraorbital notch and down across the anterior face of the bone coming out at the pyriform process to the nares or perhaps between the first and second bicuspids. In the first case, besides the impingement upon the infraorbital nerve, we have a narrowing of the external nares. In the second, there is also the introduction of a dental problem, in which the bite is poor because the upper teeth no longer match the lower. The body of the maxilla may be comminuted and crushed into the maxillary sinus, with hemorrhage. The fragmented edges are impacted into each other, or overriding. In most cases there is a fracture of the zygoma.

Since, in fractures of the face, healing takes place rapidly, because of the good blood supply and impaction, it becomes increasingly difficult to separate the fragments; therefore, it is highly important to reposition the nasal and maxillary bones as soon as shock resulting from the accident is controlled. It is usually possible, and advisable, to do this during the first week. One should not wait until the swelling has subsided, because the edema definitely hides the deformity, and the face looks worse as the swelling goes down. Palpation of the bones of the nose around the rim of the orbit and proceeding to the zygoma, together with a stereoscopic X-ray, can be relied upon to tell whether there is fracture of the maxillae, the foundation bones of the face.

METHOD OF REPOSITIONING

Since the approach of choice to the maxilla is through the inferior meatus of the nasal cavity, the nasal bridge should first be mobilized and reduced. Local anesthesia of cocaine and adrenalin will control bleeding and engorgement. Packs are placed so as to block the nasociliary nerve at the junction of the frontal and nasal bones; also, at the floor of the nose near Little's area, and at the spheno-ethmoid recess posteriorly. An injection of the subcutaneous portion at the point midway between the nasion and the inner canthus, at the in-

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ferior border of the nasal bone and at the lateral cartilaginous ala will control all pain. This injection should be of procaine 2 per cent with a trace of epinephrin (about 5 minims to 10 cc. of procaine solution). The needle should be inserted from the vestibule lateral to the ridge formed by the superior alar cartilage. A small injection of the columella is also advisable. Now, with anesthesia well in hand, and bleeding controlled by the adrenalin, one may grasp the nose with the thumbs on one side and fingers on the other and mobilize and reshape the bridge. By means of a rubber-covered clamp the septum is repositioned as necessary. Then, if it is desirable to elevate the maxilla, general anesthesia may be induced by pentothal intravenously, and an opening made in the lateral nasal wall under the inferior turbinate on the side of the depressed maxilla. This can be done with an antrum rasp, and when sufficiently enlarged, a urethral sound is inserted into the maxillary sinus and the depressed bone elevated. This will take considerable force, because of the impaction, and care should be exercised not to injure the septum or the floor of the nose by using it as a fulcrum.

In several cases coming under our observation it was impossible to reposition the maxilla through the intranasal route, so a Caldwell-Luc approach was used. An incision was made in the gingivolabial fold opposite the first and second molars and the bicuspids. The soft tissues and the periosteum were then elevated, exposing the canine fossa, and an opening made into the antrum. This makes an easier lift possible, as the normal maxilla at the prominence of the cheek is like an arch, and the point to which pressure is applied by lifting is closer to the fulcrum than it is by the intranasal route; but it also introduces the danger of depressing the teeth if the fracture has extended through the gingival ridge.

After reducing the depressed bone to good position, it is best to pack the maxillary sinus with vaseline gauze, bringing the end out through the nose so it can be removed via this route in about four or five days. The incision is sutured with silk.

The nasal bones are now splinted with dental wax stents internally, and an aluminum splint externally. This should be padded with about one-eighth inch thickness of felt to avoid damage to the skin.

Application of ice to the operated area reduces the postoperative discoloration and swelling. The wax splints are removed from the inside of the nose in 24 to 48 hours, and the external splint may be left in place for about one week.

If the canine fossa approach is used, there is a risk of infection of the face through the mouth. Consideration of this eventuality would dictate use of penicillin postoperatively.

REPORT OF A CASE

G. S., a young adult male, received a fracture of the nose and right maxilla in an automobile accident on June 27. Severe shock being present, he was treated appropriately in a local hospital, but no reduction of the facial bones was attempted.

He left the hospital with a facial deformity as in Figure 1, and presented himself at my office on July 31.

His teeth were maloccluded, the right face depressed, and the nose deflected to the left. The aeration of the nasal cavities was poor on the right. He had a paresthesia of the entire right maxillary area.

He was operated on August 5 by the technic previously described. Healing of the bones was especially firm where the impaction had taken place in the anterior surface of the maxilla. Under general anesthesia the maxilla was lifted through the Caldwell-Luc approach. A firm vaseline gauze packing was placed to support the maxilla from within. The nose was

kept in position by an internal support of dental wax and an external molded metal splint held in position with adhesive tape.

The maxillary packing was partially withdrawn on the fifth postoperative day and completely removed two days later. The dental wax was taken out on the second day; the molded metal splint was kept in place for two weeks.

Penicillin therapy was given for the first three postoperative days. The patient made an uneventful recovery and now appears as in Figure 2.

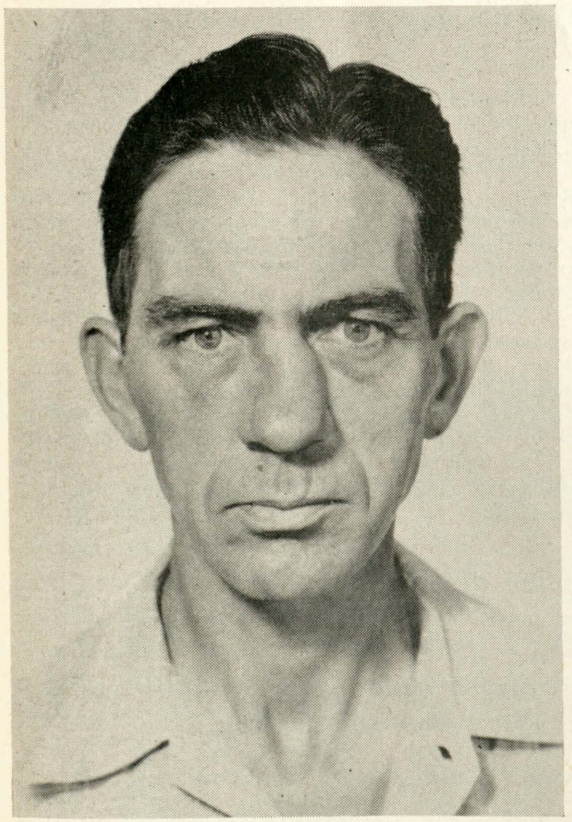

Figure 1.—Before surgery.

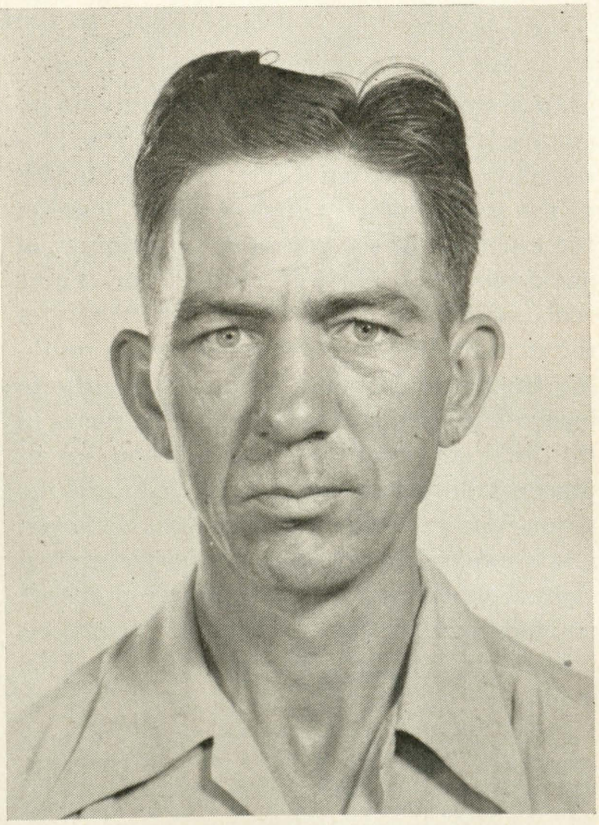

Figure 2.—As patient now appears.

CONCLUSIONS

1. Fractures of the nose and maxilla should be reduced as soon as traumatic shock will permit.

2. Adequate support of structures from within must be provided.

3. Delayed reduction results in healing of the bones in poor position, with the impacted bone being the most securely joined, and with consequent resistance to mobilization.

4. Nasal blockage may produce symptoms of paresthesia and sinusitis, which may become chronic.

CEREBRAL ANOXIA AND ITS RESIDUALS*

III. THE STRUCTURAL CHANGES

CYRIL B. COURVILLE, M.D.

In a study of the structural alterations occurring in the central nervous system it is necessary to understand certain fundamentals of the pathogenesis of such changes. Some of the essentials of the pathologic physiology of anoxia have already been pointed out. There remains to be presented a brief summary of the steps leading to the residual lesions resulting from severe oxygen want which hitherto have often been misinterpreted. The stages in the development of the ultimate lesion, for all practical purposes, may be divided into the acute, subacute, and chronic phases. However, a few important facts need to be mentioned and briefly elucidated before we are prepared to investigate the end results of the process initiated by lowering the oxygen tension of the blood.

1. The principal and most important effects of anoxemia are to be found in the central nervous system. This is due to the sensitive character of these tissues. Changes are to be found also in the lungs (thickening and cellular infiltration of the alveolar walls), the kidneys (degeneration of the renal epithelium), the liver (perivenous necrosis), the spleen (cellular infiltration), the heart muscle (brown atrophy and focal necrosis [Figure 1]), and the adrenals (hemorrhages) (Courville [1939]).

2. The immediate effects of anoxemia, as far as one can judge from the anatomic appearances of the brain and other organs, are intensive congestion and vascular dilatation, presumably resulting in a considerable degree of stagnation.

3. It is not always possible to predict the outcome (hence the extent and degree of damage to the nervous tissues) by the immediate clinical reaction. When cardiac arrest as well as respiratory failure occurs, the prognosis is usually grave. On the other hand, a fatal issue may follow even transitory respiratory failure.

4. The full extent of ultimate damage is to be seen only after an interval of several days, and progressive changes occur for a period of several weeks.

5. Clinical manifestations do not necessarily parallel evidences of physical damage to the brain. Profound manifestations may be present in the early period when there is little to be seen, and considerable recovery may occur in the presence of grossly evident lesions.

6. While some selectivity is shown in the lesions produced by asphyxia (globus pallidus and visual cortex), there is considerable variability in the extent and distribution of damage to the cerebral gray matter.

7. The exact mechanism of asphyxia has something to do with the ultimate pathologic picture, for the residual lesions in the various clinical entities show a considerable latitude of physical changes in the nervous tissues.

These factors cover the important features of the clinical course of patients who have been subjected to oxygen want, and their enumeration will serve to maintain a clinical orientation while we delve into the problems which are essentially pathologic.

CEREBRAL CHANGES IN EXPERIMENTAL ASPHYXIA

The effects of experimental oxygen want on the brain have been known for many centuries, although, to be sure, the experimenters did not always know what it was that produced the ill effects. It is recorded that during the Middle Ages traveling magicians produced temporary paralysis in goats (whose cerebral blood supply is entirely dependent upon the carotids) by firmly grasping these animals about the neck. The animal would then fall completely paralyzed, ostensibly because of some powerful mumbo-jumbo pronounced by the magician. When the grip was released, the goat promptly jumped up and ran about as before.

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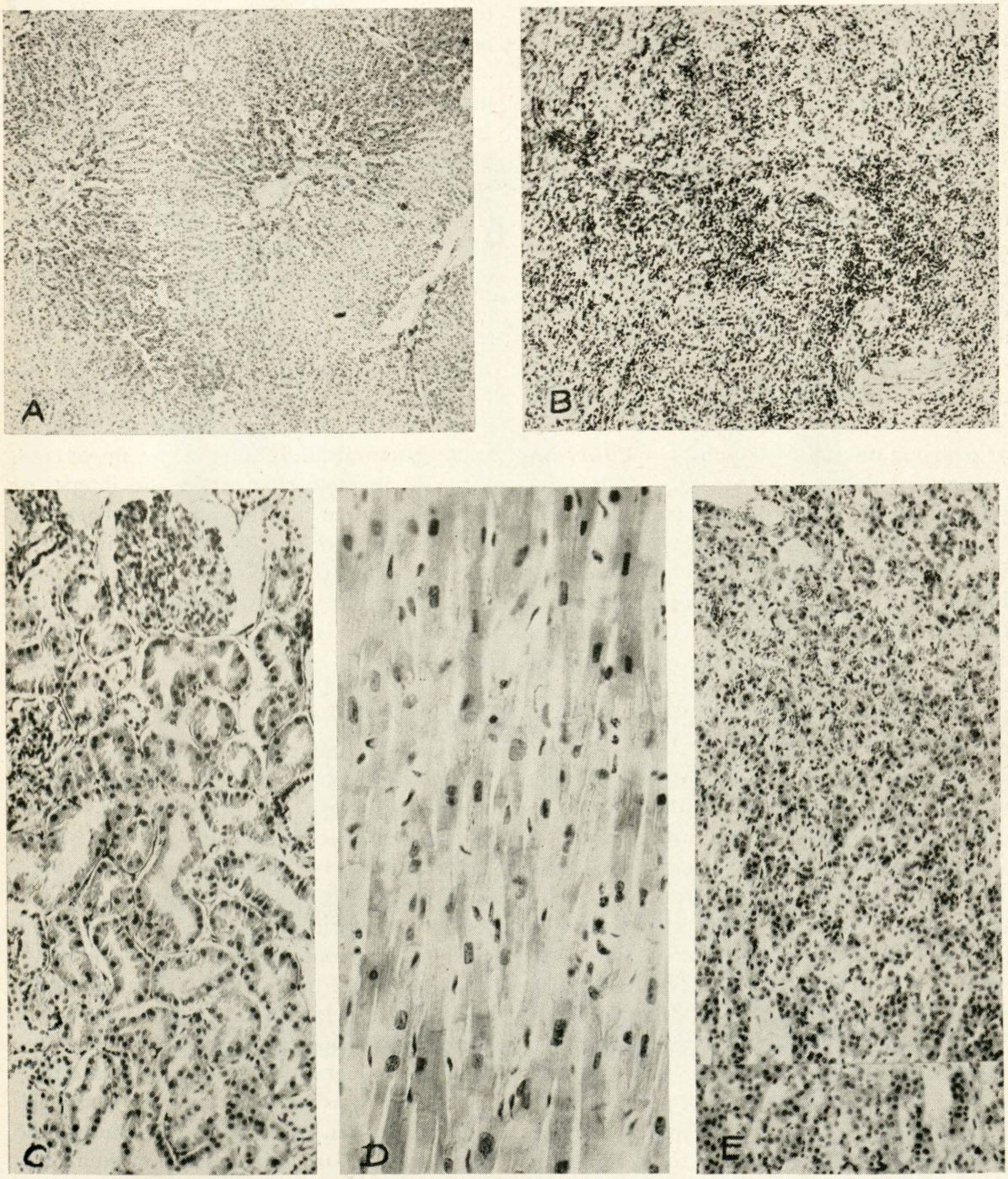


Figure 1. General pathology of anoxia. *A*. Perivenous necrosis of the liver. *B*. Cellular infiltration of the spleen (acute splenitis). *C*. Degenerative changes in the epithelium of the renal tubules. *D*. Minimal brown atrophy of cardiac muscle. *E*. Focal hemorrhages into adrenal cortex.

Sir Astley Cooper seems to have been the first to produce convulsions and other characteristic symptoms after ligature of the carotids in dogs, an experiment which was repeated by Leonard Hill (1896) over half a century later. Hill and Moot (1906) were among the first to study the alterations in the nerve cells under these circumstances. A more critical study of cerebral changes after temporary interruption of the cerebral circulation was made by Gildea and Cobb (1930), whose work remains a classic and a basic study of the effects of anoxemia.

These various experimenters have been able to demonstrate various effects on animals which are strikingly reminiscent of the effects of asphyxia on man. Convulsions—either immediate or delayed—spasticity, running fits, yowling spells, blindness, behavior peculiarities, and dementia have all been described. It is to be expected, therefore, that the changes in the brain found in animals after temporary ligation of the supplying arteries would be comparable to those found in man after profound asphyxia, since the immediate effects of ligation can be attributed only to the want of oxygen.

The histologic alterations in these animals were found to be characterized by areas of focal necrosis in the cerebral cortex, seen after a survival period of at least twenty-four hours. There was an associated dilatation of the perivascular or perineuronal spaces suggestive of some circulatory change (? edema). The nerve cells were predominantly affected, showing pyknotic change, acute swelling, ischemic change, liquefaction, vacuolization, or lipoidal degeneration. The interstitial cells were less affected, presenting evidence of reactive swelling and early proliferation. The leptomeninges were slightly thickened in some instances. In cases of death shortly after ligation the blood vessels were dilated. After an interval of time the walls of the blood vessels proved to be thickened and increased in number, and their endothelial cells contained droplets of fat.

In an interesting study of experimental neonatal asphyxia in guinea pigs Windle and Becker (1943) found that fairly typical disorders in the affected animals were to be accounted for by an absence of, or regressive changes in, the nerve cells of the cerebral cortex. These alterations are very pertinent in view of the histologic findings, to be described in later paragraphs, in human examples of asphyxia neonatorum.

If these alterations are truly typical of the condition, then we may expect to find in the human subject (1) focal necrosis, (2) predominant structural alterations in the nerve cells, with (3) reactive changes in the interstitial elements and in (4) the leptomeninges, and (5) alterations in the blood vessels indicative of circulatory changes (edema and stagnation), as well as structural changes in the vessel walls. A study of human pathology is next in order.

THE ACUTE EFFECTS OF ASPHYXIA

The immediate effects of asphyxia are variable, particularly in degree, depending upon the means by which it is produced. The most profound changes in man have been observed in balloonists who have been exposed to rarefied atmospheres. In cases of death under these circumstances the skin is a livid bluish-purple color, and hemorrhage occurs from the lungs, less often from other body apertures. Internally, hemorrhages into the brain, viscera, and linings of the body cavities are also the rule, these being the result of an intense congestion.

In cases of carbon monoxide poisoning (illuminating gas, automobile exhausts) the manifestations are much less profound. The cherry-red color of the mucous membranes and viscera is characteristic and due to the formation of carboxyhemoglobin. Hemorrhages, when present, are usually small (petechial),

but occasionally may be of larger size. Intense congestion is still the most prominent feature at autopsy.

stances may be few or absent altogether, particularly as far as the brain substance is concerned. In one of the cases studied by the pres-

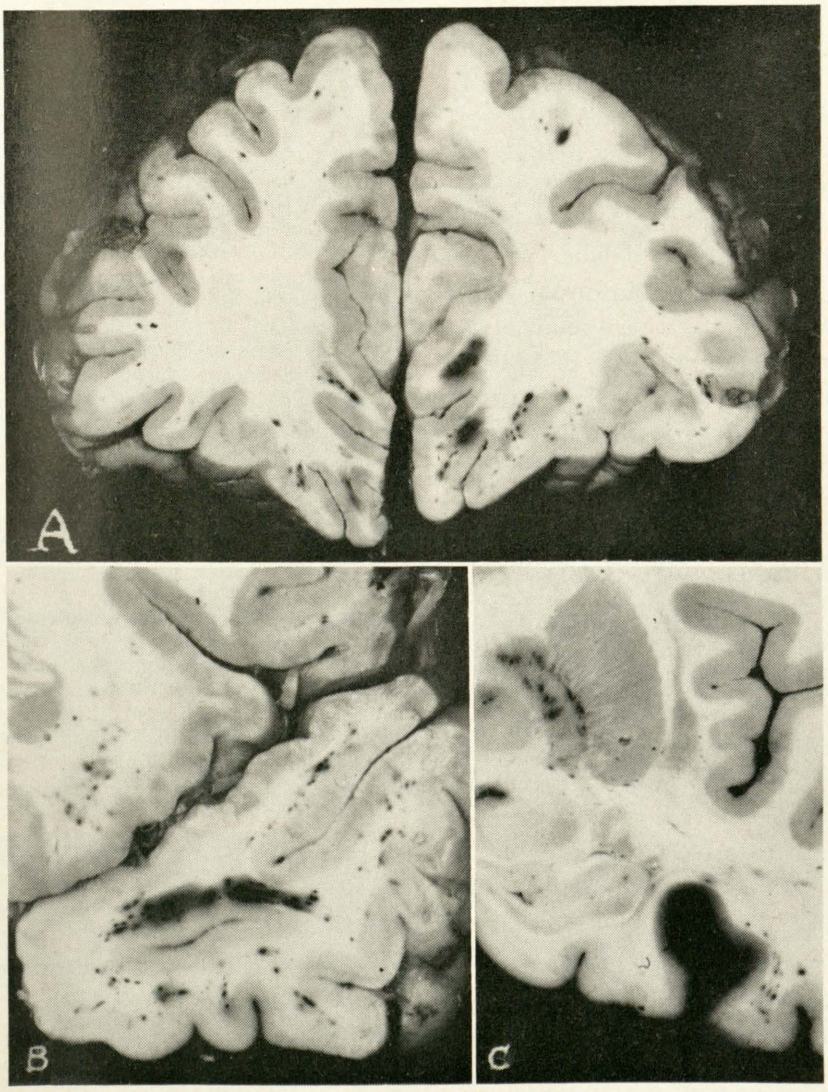


Figure 2. Hemorrhages into brain substance after asphyxia under nitrous oxide-oxygen anesthesia. Survival period six days, six hours. *A* and *B*, hemorrhages into cerebral centrum. Note tendency for hemorrhages to occur in white substance bordering gray matter. In *C* hemorrhages are found between divisions of lenticular nucleus.

Congestion is also the essential feature in cases of death after neonatal asphyxia and asphyxia after anesthesia, but it is much less profound. Hemorrhages under these circum-

ent writer there was a predilection for localization of these hemorrhages, both large and small, in the white substance adjacent to the gray matter (Figure 2).

This congestion is also apparent histologically. Sections from the various visceral organs show the small blood vessels to be widely dilated and packed with red blood cells. Small hemorrhages, perivascular in location, are often found, particularly when death comes as a result of profound and acute asphyxia.

The brain and also the blood vessels of the pia mater, the cortex and basal ganglia (less so of the cerebral centrum), and of the choroid plexus are all considerably dilated and filled with red blood cells (if the body has not been fixed by embalming before autopsy). Hemorrhages into the nervous tissue may also be found. These are the typical ball, perivascular, or ring hemorrhages indicating focal rupture of small blood vessels. Not infrequently such hemorrhages are associated with focal infarctions in which the hemorrhagic pigment, free or phagocytized, is found in the necrotic area.

The changes in the cellular elements are not striking. Acute swelling of the nerve cells with loss of Nissl's substance and acute swelling of the oligodendroglia are most characteristic.

It may be said, then, that whatever be the underlying factors resulting in damage to cells and tissues, a more or less severe degree of congestion is the rule, and the more severe the congestion the greater the likelihood of the occurrence and increase in size of the hemorrhagic effusions.

SUBACUTE STRUCTURAL CHANGES

It is in the realm of the subacute course after asphyxia that there is so much misunderstanding, both as to the clinical findings and the effects on the brain. This has arisen from the previously accepted concept that deaths due to anesthesia are immediate. That individuals may survive for a varying interval before death, or partially or fully recover from what appear to be profound insults to the brain, needs to be kept in mind.

The gross alterations in the brain in instances of subacute courses lasting a few hours to a few weeks vary considerably from one case to another; the changes are also somewhat dependent upon the means by which asphyxia was produced. In any case, it is the gray matter of the brain which is almost exclusively damaged by the process, if not entirely so.*

In instances of *carbon monoxide* (or illuminating gas) *poisoning* the resultants of asphyxia consist of areas of cortical softening and damage to the globus pallidus. The areas of softening in the cortex are sometimes of fairly large size and seem to be due to thrombosis of some of the terminal cortical arteries. The affected areas first undergo circumscribed softening, followed by depression of the area as phagocytosis of the decadent material proceeds. The subjacent white matter supplied by the vessel also undergoes softening with the overlying cerebral cortex. The globus pallidus may also undergo a type of central necrosis first manifested by a circumscribed granulation of the enclosed gray matter with subsequent liquefaction and absorption.

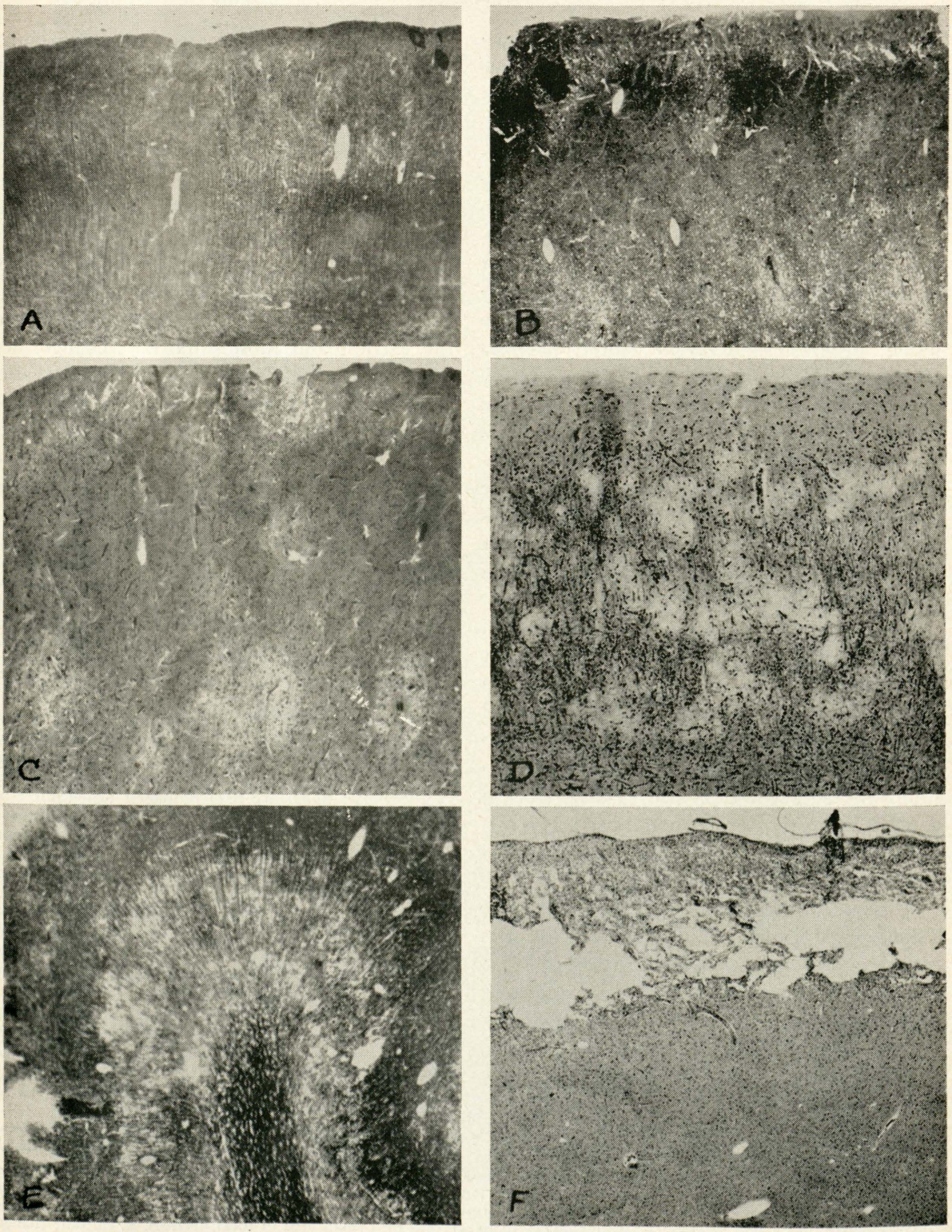
In cases of the *anesthetic asphyxias* the picture is somewhat different. No very large areas are affected, but within a few days after the episode there will be found by the palpating finger small spots of softening which suggest those following embolism. A modification of this pattern is a diffuse subtotal alteration in the visual cortex, which becomes evident grossly only on cut section. The changes in the globus pallidus are similar to those found after asphyxiation with carbon monoxide.

After *asphyxia neonatorum* as a rule death either occurs at once or else the patient survives for many months. The writer has not had the opportunity to study the brain of a case with a short survival period, nor does he know of reports of any so studied.

It is the histologic alterations within the cerebral gray matter which betray the course of the central lesions, and by following the sequence of events in the development of the cortical lesion we are able to learn something

* After experimental asphyxia in dogs, produced by exposure to carbon monoxide (Yant *et al* [1934]), degenerative changes in the white matter in the form of areas of demyelinization resulting in the formation of small cysts were noted. Similar changes were also found in the peripheral nerves. These alterations have not been found by the writer in instances of asphyxial damage to the human brain; nor does he know of such changes as may have been observed by others.

Figure 3. Development of cerebral cortical lesions in cases of anoxia. *A*. Earliest evidence of focal cortical necrosis. *B*. Multiple foci of necrosis showing beginning limitation of margins. *C*. Well-defined foci involving several cortical layers. *D*. Early fusion of individual foci. *E*. Well-defined laminar necrosis affecting entire convolution. *F*. Subtotal cortical necrosis.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of the pathogenesis of this lesion. The various stages of development of the cortical lesion are shown in the accompanying series of photomicrographs (Figure 3).

As has been suggested in the previous section, the earliest lesion to be found in the brain in cases of postanesthetic (nitrous oxide-oxygen) anoxemia consists of early degeneration of small groups of cells with enlargement of the perivascular spaces and the formation of fluid spaces in the interstitial tissues. As the lesion progresses, areas of *focal necrosis* (*Herde*) develop which prove to be simply enlargements of the areas of degenerative changes affecting but a small group of cells. As Gildea and Cobb (1930) found experimentally, these areas of devastation appear to be the basic lesion, one which seems to be characteristic of the condition. Only when this is fully recognized is one in a position to evaluate critically the ultimate lesion.

Next in order in the line of development of the cortical lesion is *zonal necrosis*. In a survey of the distribution of these lesions it has been shown (Courville [1939]) that the cortical layer involved varies from one region to another in a given case and that no one layer is uniformly affected. It is also evident from a study of interval cases that zonal necrosis is but the result of fusion of multiple areas of focal necrosis.

In turn, the fusion of these multiple stratigraphically disposed laminae of zonal necrosis results in *subtotal cortical disintegration*. In the writer's experience this advanced degree of change resulting from asphyxia alone (without the intervention of vascular alterations) is found characteristically if not exclusively in the visual cortex in the region of the calcarine fissure after nitrous oxide anoxemia.

In the globus pallidus* only two types of degeneration have been found, viz., focal necrosis and subtotal necrosis. The absence of an intervening stage of zonal or laminar necrosis is obviously due to the lack of arrangement of cells in layers with their attendant characteristic blood supply.

The alterations in the cellular elements of the brain are of special interest to the pathologist. While injury to nerve cells and fibers is the earliest and most important change, destructive as well as reactive alterations are also found in the interstitial elements, in the leptomeninges, and in the blood vessels. These changes, some of the more important of which are shown in the accompanying figure (Figure 4), deserve brief mention.

Alterations in the *nerve cells* consist in pyknotic change (sclerosis, *Zellschrumpfungen*), ischemic alteration, acute swelling, acute necrosis, pigmentary atrophy and ferrugination ("calcification"), depending upon the time interval after the asphyxial episode and the proximity of the cell areas of necrosis. These alterations in the parenchymatous elements are, after all, the most important, for they account for the clinical manifestations of the patient, whether these be acute, subacute, or chronic. A study of these changes in the parenchyma sheds considerable light on the etiology and pathogenesis of such changes as observed under other circumstances.

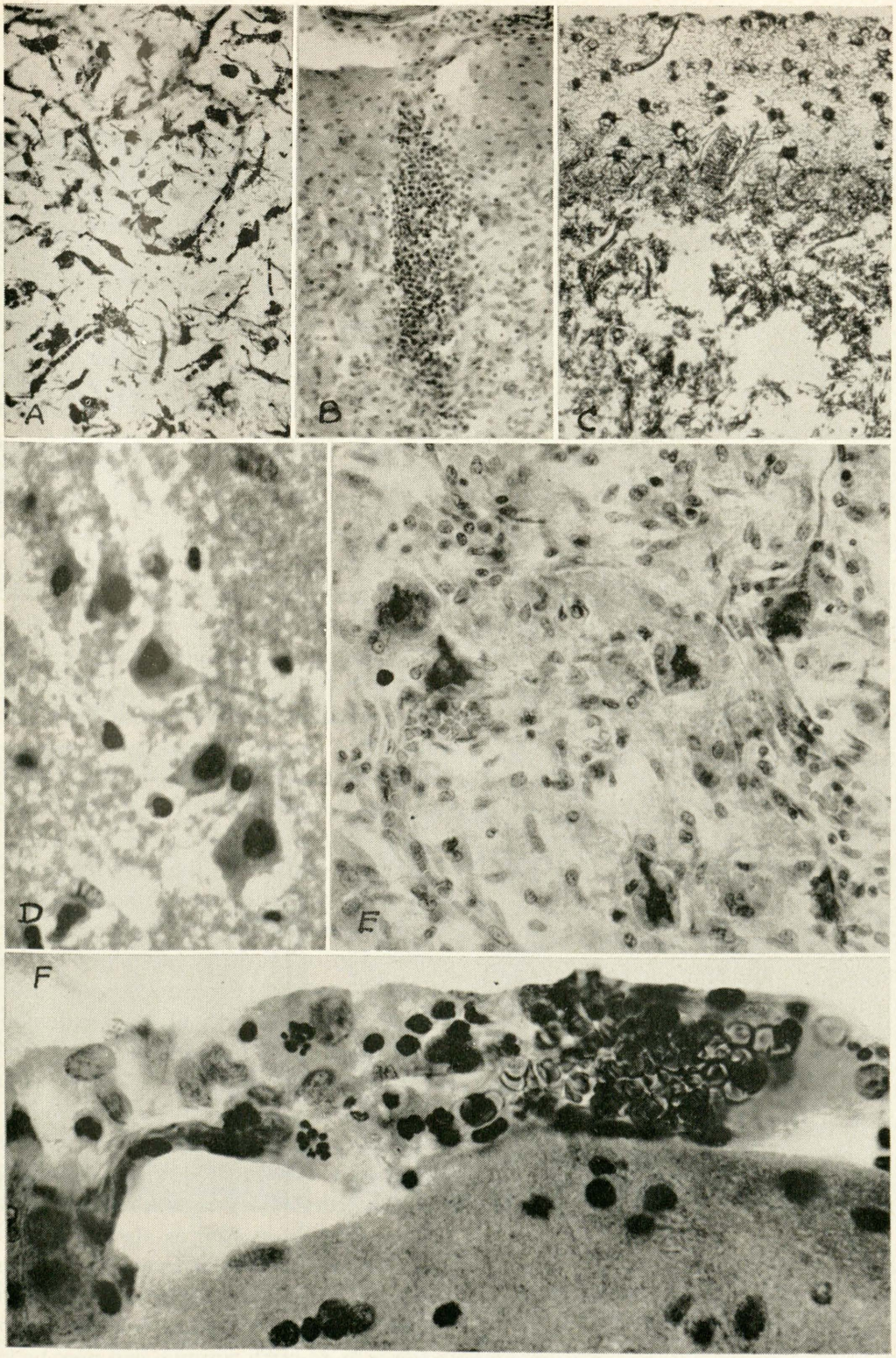
The *nerve fibers* are also damaged either directly (when adjacent to, or enclosed within, an area of focal necrosis) or indirectly, due to destruction of the parent cell. These alterations, not specific for asphyxia, involve not only the axis cylinders but also the myelin sheaths (Courville [1939]).

The supporting *astrocytic* network also suffers. Destructive changes occur in these elements within the areas of degeneration, while the glia cells bordering these areas undergo active and advanced proliferation

* The deposit of iron (calcification) in the small blood vessels of the lenticular nucleus has long been recognized as a change resulting from asphyxia, being found characteristically in individuals who succumb after exposure to carbon monoxide. The writer has studied the brain specimen in one case in which such deposits were present sixteen hours after asphyxiation. In fact, in the cases with short survival periods these vascular changes in the lenticular nucleus may be the only demonstrable abnormal findings.

→

Figure 4. Essential features of recent histopathologic alterations in the cerebral cortex after anoxia. *A*. Acute reactive changes in microglia provoked by tissue destruction. *B*. Perivascular round-cell infiltration observed three and a half weeks after an asphyxial episode. *C*. Proliferation of subpial astrocytes over subtotal destruction of the occipital cortex (interval, three and a half weeks). *D*. Acute changes in nerve cells in a small area of focal necrosis. *E*. Chronic change (ferrugination) of nerve cells. *F*. Mitoses in endothelial cells of small blood vessel leading to new vessel formation.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to form a dense glial scar. Proliferation of astrocytes is found also in the subpial region in cases of extensive cortical necrosis.

The *microglia* likewise respond promptly to degenerative changes with swelling and structural alterations leading to the formation of compound granular corpuscles. Phagocytosis by these cells is prompt and energetic, and one finds in cases of survival of three weeks or more that the debris has been cleared away to a remarkable extent.

As for the *oligodendroglia*, acute swelling of generalized distribution is the rule, with regressive changes taking place in those elements within areas of necrosis.

The *arachnoid* is found to be proliferated (chiefly the cap cells) and often adherent to the underlying *pia mater* in subacute cases. These changes appear to be the most prominent over areas of severe cortical injury.

Alterations in the *blood vessels* are important not only because of histologic interest but also because of the significance of such changes in the production of secondary lesions in the brain. Within three weeks after an asphyxial episode one finds evidence of proliferation (mitosis) of the intima of the small cortical and ganglionic vessels leading to proliferation of newformed channels. This leads to the formation of a highly vascularized scar, with secondary loss of parenchymatous elements incident to interference on a different basis from that of simple anoxemia.

In addition to the evidences of proliferation observed in the smaller vessels, there occurs a similar change which affects specifically the intima of larger ones. This results in obstruction of these larger vessels with secondary softening of larger areas of the cortex. These changes are especially characteristic of later residuals of carbon monoxide asphyxiation. It has been suspected that similar changes occur in many cases of neonatal asphyxia, leading to the formation of small cortical cysts or the larger porencephalic cysts (Courville and Marsh [1944]).

It is clearly evident that while these asphyxial alterations of cells and tissues (only outlined in this connection) may be quite technical in their aspects, it is these changes which explain the ultimate pathologic picture as well as the intercurrent clinical picture. This will be shown to be true from a pathologic viewpoint in the succeeding section of this paper, and also more fully from its clinical aspects in the one to follow.

THE ULTIMATE PHYSICAL RESIDUALS OF CEREBRAL ANOXIA

Because some patients survive an asphyxial episode for many years, and because in some instances this episode has not been correctly interpreted for what it really is (or perhaps has been entirely forgotten), the ultimate residuals in the form of physical changes in the brain have long gone misinterpreted. We still do not know what the ultimate changes in the brain are after asphyxia under anesthesia, for no known case has been followed over a period of years with final opportunity for a critical study of the cerebral tissues. The same is largely true of the carbon monoxide asphyxias. Even the residuals of neonatal asphyxia, common enough in clinical practice, have usually been mistaken for the results of traumatic lesions of the brain—assumed to be the effects of subdural, subarachnoid, or intracerebral hemorrhages. But we are now able to anticipate in some cases just what these changes may be, judging from the subacute lesions. In other instances of asphyxia of the newborn there is ample evidence of the ultimate changes, and these will now be briefly discussed.

As pointed out heretofore, the criterion for the establishment of the anoxic etiology of a given lesion of the brain is the discovery of histologic changes in the gray matter of focal character, in other words, residuals of focal necrosis. Simple recourse to a microscopic examination of blocks of atrophic cerebral cortex in many cases of spastics, idiotics, athetoids, ataxics and epileptics will serve to demonstrate these lesions. And, contrary to accepted concepts, these characteristic acellular areas will be found in cases of focal cortical scars ("microgyria"), lobar sclerosis of childhood (Friedman and Courville [1941], hemispheral (cerebral or cerebellar) agenesis (Courville and Marsh [1944]), as well as about focal cortical cysts (Penfield and Erickson [1941]) or porencephalic cysts. Many of these lesions, formerly considered to be due to imperfect morphogenesis or to birth trauma, can now be proved to be of asphyxial etiology.

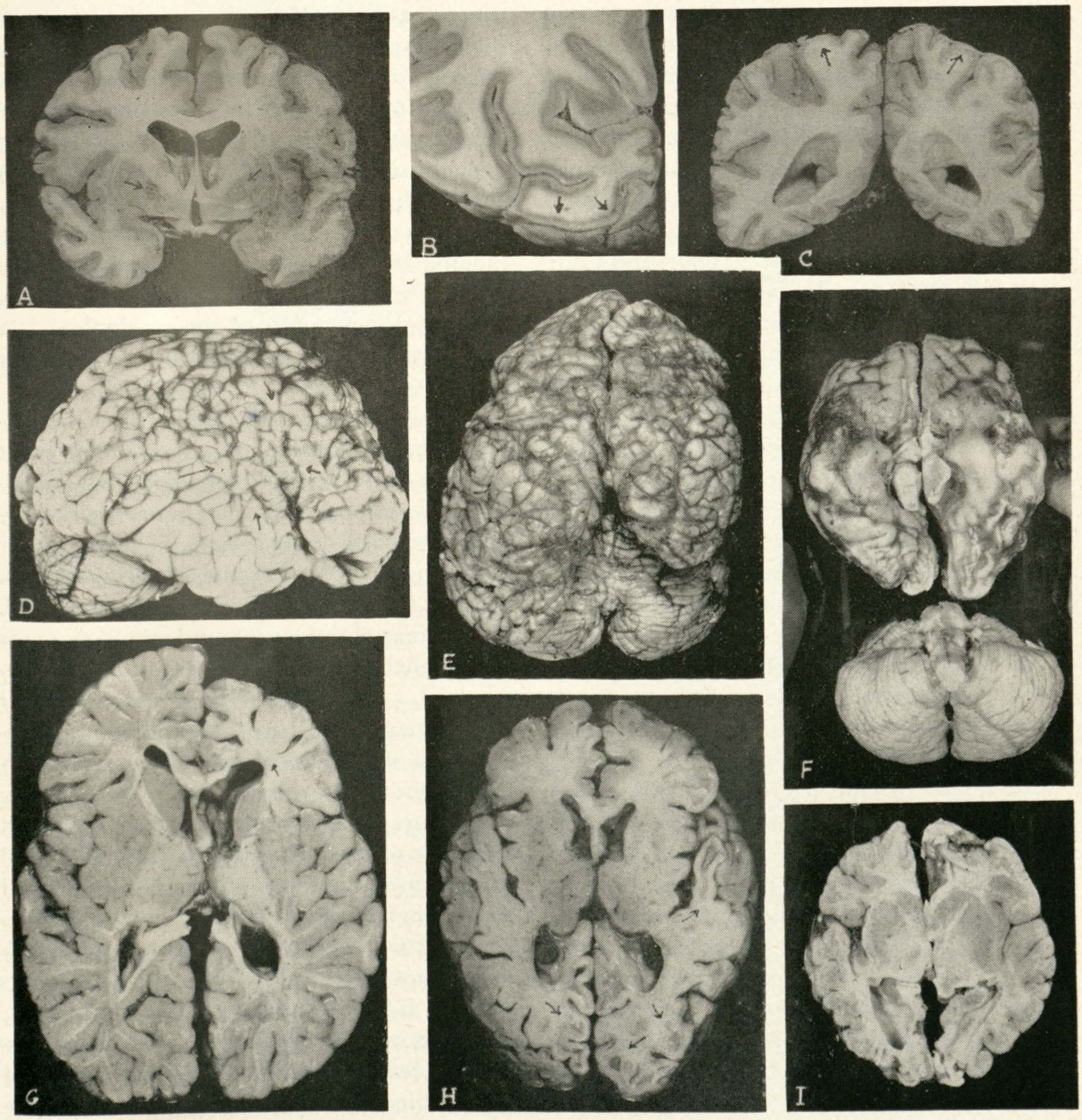

Figure 5. Residuals of asphyxial episodes of various etiologies. *A*. Degeneration of the globus pallidus after carbon monoxide "poisoning." *B*. Necrosis of the occipital cortex three and a half weeks after nitrous oxide anoxia. *C*. Softening of the parietal cortex bilaterally after carbon monoxide asphyxia. *D*. Focal cortical atrophy (lobar sclerosis, ulegyria), a residual of neonatal asphyxia. *E*. Marked hemiatrophy of the brain resulting from birth asphyxia. *F*. Profound changes in the brain of an idiotic, epileptic infant resulting from severe asphyxia at the time of delivery. *G, H, I*. Series of horizontal sections through brains showing variable degrees and distribution of changes following neonatal asphyxia.

This is but another way of saying that the ultimate residuals of neonatal asphyxia may be found in the brain in the form of focal cortical scars or cysts, atrophy of the cortex of a single lobe (ulegyria), a hemisphere, or of the entire brain with shrunken cortex (microgyria) of varying degrees as the characteristic change (Courville and Marsh [1944]). In some cases the globus pallidus of one or both hemispheres is likewise atrophic (Abbott and Courville [1938]). It has also been pointed out that instances of large porencephalic cysts are similarly to be accounted for as the result of vascular occlusion due to a proliferation of the cells of the vascular intima. These lesions have long been considered the residual of birth hemorrhage into the brain substance.

Histologically the typical finding is the loss of nerve cells in focal areas and laminae; in the extremely atrophic cortex no cells may be evident. These findings imply the selective destruction of nerve cells by oxygen want.

As for the residuals of other types of asphyxia, one cannot be so sure, for opportunities to study the brain after an interval of years is a relatively rare experience. One can only say that variable degrees of atrophy of the cortex and the globus pallidus are to be considered. This atrophy may be localized or generalized. Small vascular scars in cases of carbon monoxide asphyxia are also to be expected.

Some of the more characteristic gross changes following the anoxias of nitrous oxide-oxygen anesthesia, of carbon monoxide intoxication, and of neonatal asphyxia are shown in the accompanying illustration (Figure 5).

GENERAL CONSIDERATIONS

The story of the mechanism and effects of cerebral anoxia may now be considered to be fairly complete, at least in its larger outlines. In the case of nitrous oxide the pathogenesis of degenerative changes in the cerebral gray matter has been traced through the acute and subacute phases (Courville [1938]). Although the case in the relatively rare instances of anoxia after ether anesthesia has not been completely settled, the residual clinical findings strongly suggest a similar picture (Courville [1941]). In instances of neonatal asphyxia we lack information as to the subacute phase, but, judging from the clear-cut picture presented in the chronic cases, one can only conclude that the degeneration of the cerebral and/or cerebellar gray matter is likewise a progressive lesion (Courville and Marsh [1944]).

In cases in which the anoxia is not too profound the nerve cells of the cortex or lenticular nuclei seem to be selectively damaged, and ultimately disappear. This accounts for the primary shrinkage of the cortex, the white matter becoming atrophic because of secondary loss of nerve fibers when their parent cells are dead. In instances of more profound anoxia not only the parenchymatous elements but also the interstitial cells undergo destruction. Under these circumstances we find the subtotal destruction of the cortex, as noted in cases of survival from three to six weeks.

In those cases in which survival is limited from two to seven days the characteristic focal necrotic areas are to be found either in isolated form or in laminar arrangements. It is the presence of these focal areas of destruction which makes possible the clear recognition of the lesion, be it acute, subacute, or chronic.

But much time and space have been occupied in leading up to the clinical aspects of the problem. Since it is these clinical symptoms upon which we are dependent for a diagnosis and whose course indicates the prognosis of a given case, these matters will next be given due attention.

NOTE.—The bibliography will appear at the end of the completed article.

(To be concluded)

CANCER OF THE LUNGS

(*Continued from page 54*)

mural growth or stenosis from pressure without. Respiratory obstruction leads to atelectasis, pneumonitis, and abscess of the affected lobe. Hemoptysis occurs from erosion of blood vessels. Occurrence of infection leads to fever and toxemia. Cachexia, loss of weight, and anorexia are later symptoms. Horner's syndrome, brachial plexus disturbances, cordal and diaphragmatic paralysis are late complications.

There is a general agreement that bronchiogenic carcinoma originates from the basal cells of the epithelium of the bronchial mucous membrane. The epithelial cells of the tracheobronchial tract are undifferentiated. When carcinoma develops in the lung, the epithelial cells usually develop into one of the following: (1) squamous cell carcinoma, (2) adenocarcinoma, (3) oat cell carcinoma, (4) small cell carcinoma, or (5) transitional cell carcinoma. In 1945 Ikeda, at the University of Minnesota, described an alveolar carcinoma which differed from most pulmonary carcinoma in that it originated in the alveoli rather than in bronchial mucous membrane. It occurred with equal frequency in both sexes.

Bronchial carcinoma shows a definite predilection for the right bronchus, probably because of the more direct course and wider diameter of the right bronchus, with greater chances for exposure to various irritants.

Fully 75 per cent of bronchiogenic carcinoma originates in the main stem and primary bronchi. Thus, in three out of every four cases a bronchoscopist can visualize the growth, and remove a biopsy specimen for microscopic diagnosis. If the stem bronchus is involved, he can definitely state how close the growth is situated to the bifurcation. Direct observation of the shape of the carina, presence or absence of distortion and fixation of the bronchi, and other pertinent features are all helpful in deciding the operability of the case.

For cellular study the bronchial secretion is directly aspirated through a bronchoscope. A presumptive diagnosis of carcinoma is made if the examining bronchoscope reveals purulent or bloody discharge from the involved segmental tube even though the growth may not be seen.

X-ray is one of the most reliable single means of diagnosis, although there is no pathognomonic roentgen sign of bronchiogenic carcinoma. The density of the expanded lung is less than any other soft tissue, lending itself most admirably to X-ray scrutiny. The size of the opaque shadow is no indication of the true dimension of the growth. Stenosis results in emphysema. Complete blocking causes atelectasis of the distal lung. In every suspected case of primary bronchiogenic carcinoma a roentgenographic study should include the spine, ribs, long bones, and complete gastro-intestinal series to make certain that no metastasis is present.

No case of carcinoma of the bronchus is permanently cured by any form of irradiation. Pneumonectomy is the only known measure of salvaging the otherwise doomed patient. However, the recorded cases of five-year cures are not numerous. Operative risk of pneumonectomy as performed in well-organized hospitals is no higher than that of any other major procedure for malignancy. Sepsis is controlled by judicious use of antibiotics and sulfa drugs.

An exploratory thoracotomy offers the only certain means of proving the presence or absence of the growth, when a preoperative diagnosis cannot be established. If we are to reduce the present mortality rate from this form of malignancy, there must be concerted efforts among all of us—general practitioners, internists, radiologists, bronchologists, and thoracic surgeons.

CURRENT COMMENT

REHABILITATION OF THE DEAFENED*

LESLIE D. TROTT, M.D.

The forerunners of our American schools for the deaf were established in Europe during the year 1760. Abbe M. de l'Epee systematized the instruction of deaf mutes in his own school in France, and simultaneously Thomas Braidwood opened a private school at Edinburgh in the British Isles. Not until 1817 did the United States open its first permanent school for the deaf. A survey in the city of Hartford, Connecticut, discovered 84 deaf children, and when 400 more were found in New England, it was decided to do something about it. Funds were raised sufficient to send a theological student, Thomas H. Gallaudet, to Europe to study methods of educating the deaf. Finding the schools in London and Edinburgh closed to him, he went to France, and learned sign language and finger spelling. He returned, bringing an experienced French teacher named Laurent Clerc. Additional funds were rapidly gathered, and the Connecticut Asylum at Hartford was opened April 15, 1817, after a charter was granted by the Connecticut legislature. Henry Clay, Speaker of the House of Representatives, became interested in 1819, and succeeded in obtaining a Federal grant of twenty-three sections of wild land. It was owing to this munificent gift that the name of the school was changed to the American Asylum. Other States sent their deaf children—Massachusetts, New Hampshire, Vermont, Maine, and Rhode Island, as well as Georgia and South Carolina.

Rev. John Stanford started a private school which is now known as the New York School for the Deaf. Pennsylvania, Virginia, and Kentucky followed. Since 1843 the oral method of teaching has become accepted as the most practical. Over the years the deaf have been taught not only to communicate with the sign language but to speak and to read lips and interpret facial expressions. At the present time greater emphasis is placed on teaching the deaf to mingle with normal-hearing people in as natural a way as possible.

In 1929 a resolution framed by the principals of American schools for the deaf read as follows: "The time has come for all schools to recognize the practical value of speech and lip reading in all the activities of the schools and in all the relations of life outside the schools."

The proportion of pupils being taught orally rose from 20 per cent in 1892 to 72 per cent in 1940. Only 27 per cent were being taught to speak in 1884, whereas over 86 per cent in 1940 received instruction and could converse (though deaf to their own voices).

There is still a strong feeling among the deaf that the sign language is a beautiful one and is of priceless value. In 1930 the National Association of the Deaf affirmed "that the oral method does not give the chance for the best education, and that methods best adapted to the all-round development of the deaf child should be employed; and that the sign language appears the only practical satisfactory means by which the deaf may understand lec-

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tures and services, participate in debates and discussions, and enjoy mental recreation and culture."

The Empire State Association of the Deaf in 1938-40 published this statement: "The educated deaf bear overwhelming witness to the truth that the sign language and manual alphabet are the most practical, convenient and dependable medium of expression for those without hearing."

At a recent religious meeting I noticed a woman standing before a group in the balcony beside the rostrum. During the sermon she would translate as rapidly as the minister spoke, using her hands, fingers, arms, and facial expression. It was a magnificent demonstration of human ingenuity to hurdle the handicap of deafness. To see this instantaneous transfer of the spoken thought into visual language that others might grasp it was indeed thrilling. Only intensive attention, heaped upon hours of practice, could achieve such a harvest of accomplishment.

Over recent years it has been demonstrated that 97 per cent of the children in schools for the deaf have residual hearing. Consequently, with the advent of vacuum tubes for the amplification of sound without distortion, the possibilities are expanded for teaching with the aural method. Not that hard-of-hearing persons can ever hear like normal people, but they can be educated by sounds to differentiate between, and, most of all, to be alerted to, the vibrations of speech, music, or noise.

The present general methods of teaching may be summarized as follows:

1. Oral—speech training and speech reading.
2. Manual—sign language and digital spelling.
3. Scriptorial—writing.
4. Acoustic—aural.
5. Combined—oral and manual.

When the vocal articulation is quite unsatisfactory and hardly worth the effort, a transfer to the nonoral means of instruction should be accomplished.

Of the 64 residential schools, 45 use the combined, 12 the oral, and only six the manual alphabet method of instruction. The United States has invested $55,000,000 in the school plants, and spends $7,760,000 for maintenance of residential schools and $750,000 for day schools per year (1940-41). Private schools spend about $250,000. This makes an average cost per pupil per annum of $540 in residential schools, $250 for the day school, and over $300 for the private school. Compare this with the $88 per pupil in the common school and you see that the deaf child costs 3 to 8 times as much to educate.

"There has been produced (though the world in general knows little of it) after years of trial and discouragement and patience and determination, a system of specialized education that should be a source of pride to all who have had some part in it, a blessing to those who have benefited by it, and an honor to the civilization in which it has a place."

For the preschool child, day schools have been developed in which the mother with the child can be instructed. The John Tracy Clinic in Los Angeles, California, has added an educational program by correspondence. Recently many Australian mothers contracted rubella early in their pregnancy, and their babies were born with defective hearing. Over 25 of these have been enrolled and are receiving their lessons by mail from this clinic. Private contributions support this effort.

So far we have been considering the deaf child, who is understood to be one whose sense of hearing is nonfunctional for the ordinary purposes of life. Those deaf at birth are the congenitally deaf. Those whose deafness comes on soon after birth through disease or trauma, before the ability to speak is established, are the adventitiously deaf. Less than 100,000 compose this group, in the United States.

The children in the public schools are classi-

fied according to their hearing loss and fall into four groups, as follows:

1. Loss of 20 decibels or less in the better ear.
2. Loss of 40 decibels or less in the better ear.
3. Loss of 60 decibels or less in the better ear.
4. Over 60 decibels loss in the better ear.

Recommendations are made according to these groups, with individual variations, for example:

1. Favorable front seat.
2. Favorable front seat and include lip reading. Some speech correction when needed. Hearing aid, may or may not be used.
3. Front seat, lip reading, speech correction, and special tutoring. Hearing aid, important. Partial attendance in regular class for certain subjects. Partial attendance in special class for hard of hearing in regular school.
4. Full attendance in special class for hard of hearing in regular school. Full attendance in special day school. Full attendance in special co-educational boarding school. Hearing aid may or may not be helpful.

With the marvelous advance in hearing-aid manufacture, so that a single plastic case of small dimensions and light weight contains an entire amplifying and receiving system, it has become very practical for children to wear an aid. Here again we must give credit to the engineers and sponsors who have given to the deafened this perfection of sound reproduction in these miniature radios for inconspicuous wearing.

Over 3,000 servicemen who were deafened in World War II have been added to the millions in civilian life who find themselves unable to carry on normal conversation with their fellows. To those in adult life who become so afflicted, injurious psychological effects often develop, very frequently out of all proportion to the hearing loss—a damaged personality, which baffles the individual himself. He does not appreciate the connection between changes in his power to hear and changes in his emotional, intellectual, social, and economic life.

The sight sense informs us of the physical environment, whereas the hearing sense deals with the social environment. For contact between our mind and that of the speaker, we rely on our ears. Lacking a clear understanding of what the other person says, makes for many annoying fumbles, and perhaps many humorous ones too. No other disability yields such a crop for gag men, yet we seldom hear of any joke about the blind. As one deafened young woman factually stated, "What boy wants to shout, 'I love you!'?"

With the Medical Corps in charge of the rehabilitation of the deafened, both the Navy and the Army organized every available educational, medical, acoustic, engineering, psychological, and vocational talent. Four centers particularly concentrated on this problem. The one for the Navy is at Philadelphia, under Capt. M. J. Aston, with Commander Francis L. Lederer as head of the Otological Division. The three for the Army are: Deshon General Hospital in Butler, Pennsylvania, Borden General Hospital in Chickasha, Oklahoma, and Hoff General Hospital at Santa Barbara, Calif.

Thus were gathered together otologists, with the latest and best equipment for measuring hearing and with the finest hearing aids, acoustic physicists, technicians, psychologists, and the best-trained instructors in speech correction, hearing training, and lip reading. In addition were the therapeutists and vocational advisers. The entire nation will profit from the extensive research carried on, primarily for the war-damaged hearing of our gallant men and women. Naturally a civilian could not avail himself of such extensive service as has been accorded to these men; but the findings are being made available to adapt to civilian need.

After an enlisted man or officer is diagnosed by his local physician as eligible, he is assigned to one of the afore-mentioned hospitals. The Medical Officer becomes responsible, and outlines his needs after pure-tone audiometric, speech reception, and speech discrimination tests, as well as others, are made for diagnosis. A technician then makes appropriate individual fitting for each hearing aid instrument

(choosing ten from the council-accepted list). The patient tries each instrument for forty-eight hours. To try all of them it takes about two and one-half weeks' time, and thus he learns the limitations of an aid. The men are given hour after hour of intensive classroom drill in learning how to hear with a hearing aid, and are graded on the results obtained. For example, he must learn how to telephone, how to conduct an interview with a typewriter clattering at the next desk, how to follow a conversation with three or four people talking simultaneously in the same room, how to shut out background noises in the subway and at restaurants, how to listen to music or lectures, and how to care for his instrument and batteries.

From the three or four instruments selected by the patient, he is given further evaluation tests in the laboratory: psychoacoustic, special word lists, and tests for threshold level and tolerance limit (dynamic range of instrument), ability to hear against background noises (the instrument which allows for the greatest intensity of static is most practical). If, finally, there are two instruments which give equal efficiency, the patient makes the choice.

There are intensive courses in speech and hearing re-education for those needing them. What formerly took one and a half years has been condensed to eight weeks. That has been accomplished at the Navy hospital by a professional therapist of outstanding ability, Lt. (jg) Miriam Pauls.

We must not forget the newer approach to make speech immediately visible on the screen in the form of a sound spectrograph. This is being tested out in the Bell Telephone Laboratories. Quoting from their recent book *Visible Speech,* page 288: "Principles for the translation of speech into meaningful patterns are established, but a considerable amount of work remains before the deaf will benefit by this result even though it does constitute a major advance."

SUMMARY

The educational program for the deafened children originated in Europe and was brought to America through the stimulus of sympathetic Christian ministers and laymen. The sign language and finger spelling have given way to the more natural speech-reading and speech-using methods of today. The advent of improved hearing aids, especially the vacuum-tube type, has greatly augmented the enjoyment of social contacts for the deafened individuals. Combining all the past accomplishments and uniting the personnel for the rehabilitation of the servicemen who under shock and strain have lost their acuity of hearing, the Medical Corps have taken a splendid forward step, and have made a grand showing in results. Their research has brought to us all a better understanding of how to select hearing aids, showing their limitations and emphasizing the importance of individual application in the use of the "listener tool."

The person who needs an aid but refuses to wear one is often more conspicuous by reason of his mistakes in understanding what is said to him, or by his lack of proper voice control, than he would be with a cord around his neck and a button in his ear.

Ultimately a man's attitude toward his handicap determines the real seriousness of it. "Acknowledge your defect. The more intelligently you deal with it, the better your adjustment." In other words, a man's success or failure is predicated on his entire personality and character, not on the physical state of his ears.

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